

OSTEOSCOOP

News on current events in osteoporosis and rheumatology

Osterix regulates adult bone formation

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Osterix (Osx) is essential for osteoblast differentiation and bone formation, because mice lacking Osx die within 1 hour of birth with a complete absence of intramembranous and endochondral bone formation. Perinatal lethality caused by the disruption of the Osx gene prevents studies of the role of Osx in bones that are growing or already formed.

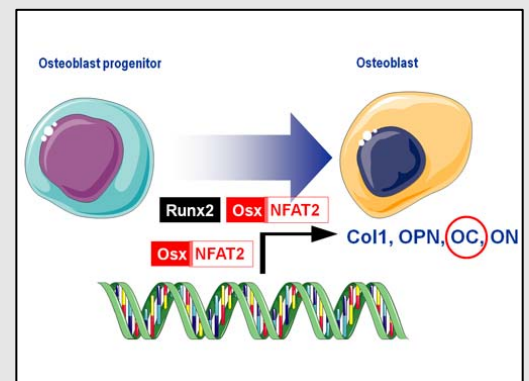
Here [1], the function of Osx was examined in adult bones using the time- and site-specific inactivation of this gene only in osteoblasts. Even though no bone defects were observed in newborn mice, Osx inactivation induced osteopenia in growing mice. BMD and bone-forming rate were decreased in lumbar vertebra, and the cortical bone of the long bones was thinner and more porous with reduced bone length. The trabecular bones were increased, but they were immature or premature. The expression of early marker genes for osteoblast differentiation such as Runx2, osteopontin, and alkaline phosphatase was markedly increased, but the late marker gene, osteocalcin, was decreased. However, no functional defects were found in osteoclasts.

In summary, Osx inactivation in growing bones delayed osteoblast maturation, causing an accumulation of immature osteoblasts and reducing osteoblast function for bone formation, without apparent defects in bone resorption. These findings suggest a significant role of Osx in positively regulating osteoblast differentiation and bone formation in adult bone.

1. Baek WY et al. *J Bone Miner Res.* 2009; 24:1055-1065.

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Differentiation of osteoblast progenitors into osteoblasts is a complex phenomenon which involves the intervention of transcription factors. Runx2 is expressed at an early stage of differentiation and controls the expression of other transcription factors. One of them is Osterix (Osx) whose importance is demonstrated by the absence of osteoblast differentiation when this factor is deleted. The expression of Collagen 1 (Col1), osteopontin (OPN), osteonectin (ON) and, above all, osteocalcin (OC), is controlled by Osterix. The lack of Osterix prevents long bones and trabecular bones from maturing.



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